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Evaluation and Prognosis of Surgical Intervention for Severe Acute Pancreatitis in Al-Diwaniyah Teaching Hospital

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Annotation: Background Acute pancreatitis can range from a mild, self-limiting process that responds to supportive care to severe disease with multiple organ failure and high mortality. Its incidence is increasing ,and pancreatitis is one of the most common causes of hospital admission for gastrointestinal illness. **Objectives:** To evaluate surgical intervention and its prognosis in severe acute pancreatitis in AL-Diwaniyah Teaching Hospital. . **Patients and methods:** A prospective study was conducted in general surgery department, in Al-Diwanyiah Teaching Hospital during the period between January 2018 and march 2021,76 patients with acute pancreatitis were admitted to the surgical ward, among them 31 patients(40.7%) were diagnosed as sever acute pancreatitis in whom 19 patients(25%) underwent surgical intervention. All patients were assessed at admission and 48 there after using the Ranson and, APACHE-II scoring system and classified according to the revised Atlanta scoring system. **Results:** From those 76 patients, 31 patients diagnosed as severe acute pancretitis(40.7%),from whom 19 patients (25%) underwent surgical intervention, among these biliary was the most common etiology (73.68%) while alcohol was (5.2%). Pseudocyst represented the most frequent indication for surgery (31.57%),followed by infected pancreatic necrosis(15.7%),while colonic perforation and bleeding were the least frequent (5.26%) for each. **Conclusion :**Multidisciplinary approach in the management of acute pancreatitis is highly recommended.Severe forms of acute pancreatitis should be managed in centers with special and sophisticated facilities if available.Surgery should be avoided in the early phases of severe acute pancreatitis except the special occasions of perforations or bleeding.Pancreatic necrosectomy for infected pancreatic necrosis with or without organ failure is a lifesaving .Intervention in the early phases of pancreatic pseudocyst involution almost doomed with recurrence.

Introduction: The pancreas is perhaps the most unforgiving organ in the human body[(1)] ,The pancreas is a retroperitoneal organ that lies in an oblique position, sloping upward from the C-loop of the duodenum to the splenic hilum. In an adult, the pancreas weighs75 to 100 g and is about 15 to 20 cm long [(1)]. Acute pancreatitis is defined as an acute condition presenting with abdominal pain, a threefold or greater rise in the serum levels of the pancreatic enzymes amylase or lipase, and/ or characteristic findings of pancreatic inflammation on contrast enhanced CT[(2)].

Pathophysiology: The underlying mechanism of injury in pancreatitis is thought to be premature activation of pancreatic enzymes within the pancreas, leading to a process of autodigestion.[(2)]The generally prevalent believe today is that pancreatitis begins with the activation of digestive zymogens inside acinar cells, which cause acinar cell injury. Studies suggest that the ultimate severity of the resulting pancreatitis may be determined by the events that occur subsequent to acinar cell injury[(3)]

Etiology: Gallstones are the most frequent cause of pancreatitis inthe Western world, in approximately 50% to 60% of patients, followed by alcohol in 20%.[(4)] The relative frequency of each of these etiologies depends largely on the population being evaluated .In both the East and the West, biliary pancreatitis is more common in women, whereas alcoholic pancreatitis is more common in middle-

aged men.[5] Other infrequent causes of acute pancreatitis are: hypercalcemia , hypertriglyceridemia, medications , hereditary causes, sphincter of Oddi dysfunction, pancreas divisum, and infections[4] .

Diagnosis and severity assessment of acute pancreatitis

Clinical diagnosis of acute pancreatitis is based on patient symptoms,

physical examination, laboratory analysis, and radiological data. According to practice guidelines published in 2006, a diagnosis of acute pancreatitis requires two out of three main features:

(1) Abdominal pain typical for acute pancreatitis.

(2) Serum amylase and/or lipase greater than or equal to three times the upper normal limit.

(3) Evidence of acute pancreatitis on computed tomography ,contrast enhanced CT and less commonly, magnetic resonance imaging (MRI)

or trans-abdominal ultrasonography[6].

Because of large variability in the clinical course of acute pancreatitis, a number of predictive scoring systems have been developed. These scoring systems are based on clinical and biochemical parameters: for example, the Ranson , APACHE-II, Imrie, or modified Glasgow scores. Blood levels of C-reactive protein and blood urea nitrogen are also often used in predicting severity at the time of hospital admission. All these scoring systems have their own strengths and limitations, and a system with a high negative predictive value and a high positive predictive value is not yet available, as described in a recent systematic review [7]

Scoring Systems in Acute Pancreatitis

Cutoff for Predicted Severe Acute Pancreatitis

1. APACHE II ≥8 in first 24 h*

2. BISAP ≥3 in first 24 h

3. Modified Glasgow (or Imrie) ≥3 in first 48 h

4. Ranson ≥3 in first 48 h

5. Urea at admission >60 mmoL/L

6. C-reactive protein >150 U/L in first 72 h

Evidence of present or evolving organ dysfunction defined as follows

1**. Respiratory**(PaO2/FiO2 ≤ 300 or respiratory rate > 20 breaths per min).

2**. Cardiovascular** (hypotension despite aggressive fluid resuscitation [systolic blood pressure (sBP) < 90 mm Hg off of inotropic support or drop of sBP> 40], need for vasopressors [not fluid responsive], or pH < 7.3).

3**. Renal** (≥ 1.5-fold increase in serum creatinine over 7 d, increase of ≥ 26.5 μmol in serum creatinine over 48 h, urine output < 0.5mL/kg/h for ≥ 6 hours ,and/or the need for aggressive, ongoing fluid resuscitation defined as evidence of severe hemoconcentration (hemoglobin [Hb] > 160, hematocrit [HCT] > 0.500)[10].

4. **Central nervous system failure** if the Glasgow coma score was <13.

5. **Coagulopathy** if platelet count was ⩽80×109/l[11].

Patients with 1 or more of the above criteria and a body mass index (BMI) above 30 (or BMI > 25 in Asian populations) should be monitored carefully, with a lower threshold for transfer to a monitored unit given the worse course of disease in the obese patient population[10].

Phases of Acute Pancreatitis:

Traditionally, acute pancreatitis was described as running a biphasic course with two peaks of mortality: early and late. The early phase is characterized by a SIRS and lasts about 1 to 2 weeks. The late phase is characterized by a compensatory, anti-inflammatory response syndrome (CARS), which can run a protracted course from weeks to months. More recent data suggest that the biphasic course is outdated and that there are not two peaks in the incidence of organ failure and mortality[14].

Laboratory tests:

1. In the setting of characteristic abdominal symptoms and/or characteristic imaging, serum levels of amylase or lipase 3 times the upper limit of normal secure the diagnosis[12].

2. Other laboratory tests, such as trypsinogen activation peptide and trypsinogen-2 levels, have been shown to be more specific than serum amylase or lipase levels,

but these tests are not readily available [12].

Imaging investigations

1. **Ultrasonography** is the initial investigation of choice in patients with jaundice to determine whether or not the bile duct is dilated, the coexistence of gallstones or gross disease within the liver such as metastases[15].

2. **Cross-sectional imaging**, particularly contrast-enhanced computed tomography (CT), plays an essential role in evaluation of the progression to severe acute pancreatitis with associated complications CT findings of simple edematous pancreatitis include enlargement of the pancreas with loss of peripancreatic fat planes, areas of decreased density, and occasional simple fluid collections The Balthazar scoring system and other similar grading systems incorporate imaging findings such as pancreatic inflammation and peripancreatic collections in an attempt to correlate radiographic appearance with morbidity and mortality[16,17].CT is particularly useful in its ability to demonstrate pancreatic necrosis ,Various criteria used to diagnose necrosis include nonenhancement of more than 30% of the pancreatic parenchyma or an area greater than 3 cm of the pancreas that does not enhance[18].The sensitivity for identifying pancreatic necrosis using contrast-enhanced CT approaches 100% after 4days from diagnosis[19].

3**. Magnetic resonance imaging (MRI)** is sometimes used as an alternative in patients with moderate renal impairment or an allergy to IV contrast .MRI may have comparable sensitivity and specificity to CT for diagnosis of severe acute pancreatitis [20].

Treatment:

Early supportive measures:

1. Pain Management: In the vast majority of acute pancreatitis patients, intense abdominal pain is the presenting symptom in the emergency department A specific pain treatment regimen for acute pancreatitis is not available, so use of the World Health Organization (WHO) analgesic ladder is recommended[21].

2. Fluid Therapy : Extensive fluid resuscitation is often needed during the first days of acute pancreatitis to correct or preferably prevent intravascular hypovolemia and maintain microcirculation of the pancreas[22,23].The recent update of the IAP/APA(International Association of Pancreatology/American Pancreatology Association) treatment guideline for acute pancreatitis recommends the use of Ringer lactate with an infusion rate of 5 to 10 mL/kg/h until resuscitation goals are reached, monitored by vital parameters and urine production[24,25].

3. Antibiotics: An extensively debated issue is the use of prophylactic administration of antibiotics to prevent infection of necrosis in acute pancreatitis. The most recent systematic reviews of randomized trials have shown that prophylactic administration of intravenous antibiotics does not prevent infection of (peri)pancreatic necrosis[26,27].

4. Nutrition: When oral nutrition is not tolerated, enteral or parenteral nutrition should be started .In predicted severe acute pancreatitis, a head-to-head comparison of enteral nutrition through a nasoenteric feeding tube and parenteral nutrition showed that enteral nutrition was superior in terms of reducing organ failure, infected necrosis, and even mortality[28].The underlying mechanism for the beneficial effect of enteral nutrition could be reduced gut permeability and less bacterial overgrowth with bacterial translocation. This may have a positive influence on intestinal motility and may help to conserve or restore bowel mucosa[29,30]. Enteral nutrition is only recommended when an oral diet is not tolerated during the first 3 to 5 days

of acute pancreatitis[31].

5. Role of Cholecystectomy: The timing of cholecystectomy in patients with gallstone pancreatitis has been debated for many decades[31],early cholecystectomy just before discharge, when the patient has recovered and severe disease excluded compared to interval cholecystectomy, effectively reduces the rate of recurrent gallstone-related complications in patients with mild biliary pancreatitis, with a very low added risk of complications[32].Early cholecystectomy should be performed after mild biliary pancreatitis has resolved. Evidence on the timing of cholecystectomy in severe pancreatitis is scarce. Cholecystectomy is recommended after all signs of pancreatic necrosis have been resolved or if they persist more than 6 weeks[33,24].

6. Role of ERCP: Patients are selected for ERCP predominantly based on whether evidence exists for obstructive choledocholithiasis. The need for ERCP in the setting of biliary obstruction and cholangitis is paramount .ERCP is not indicated in the absence of jaundice, with evidence of choledocholithiasis with a dilated bile duct on imaging, in cases of mild acute gallstone pancreatitis, or as a diagnostic test before cholecystectomy[34]. High rates of recurrence in patients with gallstone pancreatitis discharged without cholecystectomy have led to the use of ERCP and sphincterotomy for risk reduction .One prospective study showed a decrease in risk of recurrent gallstone disease from 37% to 0% with ERCP and sphincterotomy[35].

Interventional and Surgical management :

Local complications identified on CT scan include peripancreatic fluid collections, acute necrotic collections (ANCs), pseudocysts , and walled-off necrosis. The role of conservative management and minimally invasive approaches is now better defined, and more patients are able to avoid traditional surgical debridement with the use of percutaneous catheter drainage and endoscopic techniques ,No universally accepted algorithms exist, although evidence-based consensus continues to develop[36]**.**

Surgical debridement:

Surgery has involved removing necrotic pancreatic and peripancreatic tissue, preserving viable pancreas, and allowing drainage for an expected pancreatic fistula. Methods include debridement with closure over drains, debridement with open packing of the pancreatic bed or debridement with closure over irrigation drains[37,38].When open surgical debridement is indicated, exploration may be initiated with a bilateral subcostal or midline incision . The pancreatic bed may be approached via the gastrocolic ligament or through the transverse mesocolon . An approach through the mesocolon may avoid the dense inflammatory process obscuring planes between the stomach and transverse colon[39].Pancreatic debridement is accomplished bluntly, using finger dissection to remove necrotic tissue that easily separates from surrounding structures. Overzealous removal of tissue can result in hemorrhage. All fluid and tissue is sent for aerobic and anaerobic culture. Exposure and removal of all tissue may require access to both paracolic gutters, the pararenal spaces, retro-peritoneum into the pelvis, or the gastro-hepatic omentum. Surgical debridement can be followed with closed-suction drainage[37].continuous closed lavage[40],or marsupialization of the pancreatic bed with open drainage and repeat packing in patients with severe necrosis[41].In all cases, surgical therapy is delayed as long as possible, which may facilitate atraumatic debridement [42].

Minimally invasive debridement: Laparoscopic approaches to pancreatic debridement are well described and may be more successful in removing all necrotic material compared to other minimally

invasive methods[43].Advantages of this approach include minimizing wound complications, although it carries some risk of further peritoneal infection with pneumoperitoneum. It is recognized that these are technically challenging laparoscopic procedures, perhaps limiting their overall utility[44].

Direct Endoscopic Necrosectomy Endoscopic debridement is increasingly recognized as an alternative to open surgical debridement, although its availability is limited to specialized centers. Retroperitoneal endoscopy via transgastric fenestration allows direct visual access to retroperitoneal collections[45].

Percutaneous Catheter Drainage: Percutaneous catheter drainage may be useful in patients deemed unfit for surgical intervention or to address residual collections after surgical debridement. Catheters are placed under CT or ultrasound guidance, with a transperitoneal or retroperitoneal approach. Multiple catheter may be required, and repeat procedures to place new or larger catheters up to 30 Fr may be needed[46].

Prognosis: Acute pancreatitis is classified according to its morphology into edematous pancreatitis and necrotizing pancreatitis. Edematous pancreatitis accounts for 80–90% of acute pancreatitis and remission can be achieved in most of the patients without receiving any special treatment. Necrotizing pancreatitis occupies 10–20% of acute pancreatitis and the mortality rate is reported to be 14–25%. The mortality rate is particularly high (34–40%) for infected pancreatic necrosis that is accompanied by bacterial infection in the necrotic tissue of the pancreas[47].On the other hand, the mortality rate is reported to be 0–11% for sterile pancreatic necrosis which is not accompanied by bacterial infection[48].

Patients and methods: During the period between January 2018 and March 2021, a prospective study was conducted in the general surgery department in Al-Diwaniyah Teaching hospital where 76 patients with acute pancreatitis were admitted to the surgical ward, among them 31(40.7%) patients were diagnosed as severe acute pancreatitis in whom 19 patients(25%) underwent surgical intervention. All patients were assessed at admission and 48hours there after using the Ransons and APACHE II scoring system and classified according to the revised Atlanta scoring system. All patient where evaluated by a detailed history and clinical examination and investigations.

1. Complete blood picture and hematocrit. 2.CRP (C-reactive proteins). 3.Serum levels of Amylase and Lipase enzymes. 4.Liver enzymes: including aspartate aminotransferase (AST),Alanine aminotransferase (ALT), Alkaline phosphatase (ALP), and total serum bilirubin.5. Serum electrolytes and serum calcium levels. 6.Random Blood Sugar (RBS). 7.Blood Urea Nitrogen(BUN) and Serum Creatinine.8. Serum lactate dehydrogenase (LDH).9.Arterial Blood Gas analysis. 10.Abdominal ultrasound . 11.Native and Contrast enhanced CT scan.

Multidisciplinary team management were applied to most patients with severe acute pancreatitis including physician, surgeon and intensive care physician. Immediate supportive treatment with aggressive intravenous fluid therapy, central venous catheterization, bladder catheterization, blood transfusion in anemic patients, vitamin K supplement in jaundice patients, together with fresh frozen plasma infusion when indicated. Those patients who needed surgical intervention were explored by generous midline laparotomy incision surgery was performed by a senior surgeon. The type of surgical procedure was selected to match specific indication. After surgery patients were transferred to the intensive care unit. Data were collected and managed by the SPSS software 21 version. Data were tabulated and analyzed using the SPSS software and results expressed in chi square ,Odd Ratio and Confidence Interval.

Results and discussion: Seventy six patients (76) were included in this study diagnosed as acute pancreatitis among them 31 patients (40.7%) were diagnosed as severe acute pancreatitis . From these 19 patients(25%) underwent surgical intervention for complications associated with severe acute pancreatitis. Among many indications pseudocyst was the most common, other indications involved infected necrosis and abscess and exploratory laporatomy.

Table 1.Demographic characteristics

Age	20-30	30-40	40-50	50-55	55≥	Total	percentage
Male	2	3	3	2	2	12	(15.7%)
Female	0	4	2	1	0	7	(9.2%)
Total	2	7	5	3	2	19	(25%)

Table 1 showed that male are more frequent than females (with 1.7 :1 male to female ratio) to be affected by a more severe form of acute pancreatitis , Although acute pancreatitis is more common in females owing to the increased frequency of gallstones reaching a frequency of (70-80) percent of all acute pancreatitis etiology [49,50]. Males are more susceptible for more severe forms of acute pancreatitis[50,51]. and hence the greater chance for complications, other explanation is that male patients tend to present late with the consequences of delay in initiation of treatment especially in the course of gallstones disease these findings are consistent with a study by Pezzilli R, Billi P, Morselli-Labate AM (1998) [50] .

About 63 % of patients aged 30-50 years, these results were slightly different from estimate around the world , as the global prevalence increased with age up to 60–64 years and 44–49 years in females and males, respectively[52]. However there was no significant difference in the age- gender matched prevalence p value 0,1 .

Table 2. prognostic index at admission

APACHEII < 8	APACHEII >8	RANSONS < 3	RANSONS > 3	TOTAL
45	31	45	31	76

31 patients were classified as severe acute pancreatitis and managed at Intensive Care Unit by multidisciplinary team, among them 19 patients developed local complications and surgical intervention during their disease course.

Table 3. complications of severe acute pancreatitis

Local	Number	Percentage	Systemic	number	Percentage
Acute fluid collection	8	25.8%	Renal	7	22.5%
Bleeding	1	3.2%	Respiratory	5	16%
Perforation	1	3.2%	Cardiovascular	5	16%
Pancreatic necrosis	3	9.6%	Hepatic	1	3.2%
Infected pancreatic necrosis	3	9.6%	Multi-organ failure	12	38.7%
Acute necrotic fluid collection	2	6.45%	Total	30	
Pseudocyst	6	19.3%			
Total	20				

Table 3. showed the types and frequency of local and systemic complications of severe acute pancreatitis.

Local complications :

In presented study acute peripancreatic fluid collection was the most common local complication 8 cases (25.8% of total 31 cases of severe acute pancreatitis , a finding which is consistent with a study done by Xiao Dong Xu et al.(Acute Pancreatitis Classifications: Basis and Key Goals) which demonstrated acute fluid collection as the most frequent local complication[53,54].

Pancreatic pseudocyst was the second most encountered local complication 6 cases out of 31 (19.3%) which is slightly different from the above mentioned study of Xiao Dong Xu et al. which demonstrated 6.4% of pseudocyst frequency among severe cases. Pancreatic necrosis and infected pancreatic

necrosis reported in 3 cases for each (9.6%), while abscess in 2 cases (6.45%). Perforation and bleeding were the least common complications 1 case for each(3.2%).

Systemic complications:

multi-organ failure was the most common, occurred in 12 cases out of 31 (38.7%) finding which is consistent with a study done by D D Tran et al.(Acute renal failure in patients with acute pancreatitis: prevalence, risk factors, and outcome)[55]. while isolated renal organ failure reported in 7 cases out of 31 (22.5%) ,a slightly different from the same above mentioned study which documented isolated renal failure in only 2% of cases this might be explained by the fact that Our study included only those cases with severe pancreatitis requiring surgery , and isolated renal failure may constitute the majority of systemic complications in all cases of pancreatitis from which only severe forms were selected to study .isolated hepatic organ failure was the least common single organ failure in our study.

Respiratory and cardiovascular complications presented 5cases for each (16%) of patients with severe acute pancreatitis.

Table 4. Etiology of severe acute pancreatitis

Etiology	Number	Percentage
Biliary	14	73.6%
Iatrogenic	2	10.5%
Alcohol	1	5%
Others	2	10.5%
Total	19	100%

Table 4 showed represent the etiology of severe acute pancreatitis patients who underwent surgical intervention.

Our data revealed that biliary pancreatitis remain the most common factor in the etiology of acute pancreatitis with an incidence of 73% (14 cases out of 19) which is consistent with global and a study by Enas Ahmed Reda Alkareemy et al.(Etiology, clinical characteristics, and outcomes of acute pancreatitis in patients at Assiut University Hospital) in Egypt [56].

In presented study iatrogenic causes of acute pancreatitis were restricted to cases of post ERCP pancreatitis especially after common bile duct stone extraction however the actual number of post ERCP pancreatitis exceeds this figure and not included in our series because most of them either subclinical or mild form of pancreatitis managed conservatively.

Moreover alcoholic pancreatitis was the least common 1 case (5%) of surgical patients, however most of the published series listed alcohol consumption as the second most common cause of acute pancreatitis[56], this difference may be related to the lower rate of alcohol consumption or to the denial of alcohol intake, a question is not yet answered.

Table 5. Indications of surgery in severe acute pancreatitis

Indication	**Number**	**Percentage**
Pancreatic necrosis	**1**	**5%**
Infected pancreatic necrosis	**3**	**15.7%**
Pancreatic necrosis and MOF	**2**	**10.5%**
Pancreatic abscess	**2**	**10.5%**
Perforation	**1**	**5%**
Bleeding	**1**	**5%**
Pseudocyst	**6**	**31.5%**
Exploratory laporatomy	**3**	**15.7%**
Total	**19**	**100%**

Table 6. Type of Surgery

Type of complication	Type of surgery	Number of cases	Percentage
Psudocyst	**Cystogastrostomy With or without cholecystectomy**	**4**	**21 %**
Psudocyst	**External drainage**	**1**	**5.26 %**
Pancreatic abscess	**Drainage**	**2**	**10.5 %**
Infected necrosis	**Necrosectomy and drainage**	**3**	**15.7 %**
Extensive pancreatic necrosis and MOF	**Necrosectomy and drainage**	**2**	**10.5 %**
Pseudocyst and pancreatic necrosis	**Necrosectomy cystogastrostomy and cholecystectomy**	**1**	**5.26%**
Bleeding	**Packing and drainage**	**1**	**5.26 %**
Perforated colon	**Colostomy and drainage**	**1**	**5.26%**
Doubtful diagnosis	**Exploratory laparotomy**	**3**	**15.7 %**
Recurrence	**Roux En Y Cystojejunostomy With cholecystectomy**	**1**	**5.26%**

Table 6. showed the type of surgery selected for each type of complication

In presented study ,pancreatic pseudocyst was the most common indication for surgery 6 cases (31.5%), surgery for pseudocyst was performed for symptomatic large (more than 6 cm), mature cyst after 6 weeks from the onset of acute pancreatitis which fail to resolve , cysts which are immature and developed early in the course of pancreatitis were left for spontaneous resolution[57]. Recently most series recommend non interventional observation for asymptomatic non complicated pseudocyst and reserve surgery for very large , symptomatic, or complicated pseudocyst despite the widely known surgical opinion that a cysto-gastrostomy is required when a pseudocyst of greater than 6 cm and present for more than 6 week[58].

Five cases were managed by cystogastrostomy ,one case was treated by external drainage which was attempted earlier in the course of the development of psudocyst, which doomed with recurrence of the cyst and symptoms after few weeks ,the site of the cyst involved the body and tail of the pancreas, a second operation was done after 8 weeks by Roux en Y cystojejunostomy , which successfully decompressed the cyst and relieved the symptoms.

The remaining 5 cases were managed by cystogastrostomy , owing to the common usual position of the cyst lying in the lesser sac and adherent well to the posterior wall of the stomach.

In our study, surgery for pancreatic necrosis was the second common indication , we have 6 cases treated by necrosectomy , among them there were 3 patients with radiologically proved retroperitoneal gas with deterioration of clinical condition, upon which the decision of surgery was taken. Other 2 patients had an extensive pancreatic necrosis (more than 50% of the gland) with organ failure, in those patients the decision of surgery was lifesaving in spite of the absence of CT evidence of retroperitoneal gas and the unavailability of CT guided FNA. Lastly the 6th patients underwent pancreatic necrosectomy in conjunction with gastrocystostomy for large symptomatic pseudocyst. In fact recently, there were a lot of changes in the management of pancreatic necrosis owing to the development in the interventional radiology which aids both in the diagnosis and subsequent management. Secondly the indications for necrosectomy for sterile pancreatic necrosis in the absence of other complications are weaning[58].

In our study we still adopted the older method for the management of pancreatic necrosis i.e necrosectomy for nonviable pancreatic tissue , and the reasons behind that were the non-availability of interventional radiology to be engaged in the management in form of needle aspiration for pancreatic necrosis to exclude the existence of infection and no single case of pancreatic necrosis managed radiologically for the mentioned reason. Instead we totally depended on the clinical and CT features of associated infection and biochemical parameters before embarking upon surgery for pancreatic necrosis.

Open necrosectomy is most performed using a pancreas preserving technique with gentle finger blunt debridement of demarcated non-viable tissue (‘pancreatic sequestrum) with the avoidance of formal pancreatic resections and a reduced risk of bleeding, fistulae and avoiding the removal of viable pancreatic tissue[59].

Open necrosectomy is no longer considered the standard of care for the management of pancreatic necrosis . Less invasive techniques have been developed and implemented and these have largely replaced the need for open procedures[60]. Most of the centers dealing with patients with infected pancreatic necrosis has shifted from the conventional open surgery toward the less invasive procedures of necrosectomy , among them percutaneous, laproscopic, and transgastric necrosectomy each procedure require a lot of expertise , and has its own advantages and disadvantages. The success rate in terms of mortality and morbidity as compared to the conventional surgery is still debatable[61,62,63].

Massive intra-abdominal hemorrhage was documented in one patient who presented with severe haemorrhagic pancreatitis in whom the bleeding was generalized ooze from severe pancreatic necrosis due to formation of microaneurysms in the peripancreatic tissue and was managed by aggressive resuscitation and exploratory laporatomy , ligation of bleeders and packing and drainage of the pancreatic bed [64].

One case of acute necrotizing pancreatitis presented with perforation of transverse colon, albeit rare but documented complication presented with frank fecal peritonitis which necessitate immediate action, otherwise mortality would be inevitable. Very few documented cases of colonic perforation as a result of colonic necrosis secondary to severe acute pancreatitis[65]. However, a more frequent scenario of colonic perforation is the invasion by large pancreatic pseudocyst as declared by many literatures[66].

In presented study 3 cases were explored by median laparotomy for presumptive diagnosis of peritonitis from a perforated viscus , this event is no longer a proper choice in the management of acute pancreatitis and reflects a surgical mismanagement due to undue urge to operate or the poor unyielding biochemical and radiological interpretations[67] .

Pancreatic abscess results from liquefaction of limited necrosis followed by infection , it usually develop late in the course of acute pancreatitis and it has less grave sequel than infected pancreatic necrosis, recently percutaneous drainage under radiological control with appropriate antibiotic cover is the most acceptable treatment[68].Our case of pancreatic abscess was managed by conventional laparotomy and drainage .

Table 7. Morbidity and Mortality

Morbidity	Number	Percentage		Number	Cause	Percentage
Wound infection	2	10.5%	Mortality	2	MOF multi-organ failure	10.5 %
Burst abdomen	1	5.26%				
Incisional hernia	1	5.26%				
Deep venous Thrombosis	1	5.26%				
Pulmonary atelectasis	3	15.7%				

Table 7 . showed The overall mortality and morbidity in post- operative patients.

The overall mortality in our study was 2 patients out of 31 cases of severe acute pancreatitis (6.4%) ,(10.5%) from total 19 surgical patients which is comparable with a study by B Gloor et al. which documented 4% overall mortality, and 9 % for patients with necrotizing disease, these findings are different from a study done by Vesna Bumbasirevic et al. which demonstrated (53.6%) overall mortality among patients with severe acute pancreatitis in ICU[69,70]. However mortality figure in our study does not take in account all cases of pancreatitis in our center, furthermore , if we calculate the mortality in cases of infected pancreatic necrosis, the figure will rise to 33.5%, again this figure is very variable among centers depending on the available facilities and expertise. There has been great improvement in knowledge of the natural course and pathophysiology of acute pancreatitis over the past decade. The clinical course of acute pancreatitis varies from a mild transitory form to a severe necrotising disease[71].In general, severe pancreatitis develops in two phases. The first two weeks after onset of symptoms are characterized by the systemic inflammatory response syndrome (SIRS). In parallel, pancreatic necrosis develops within the first four days after the onset of symptoms to its full extent[72].Recently the mortality from acute pancreatitis including its severe necrotizing form which is mainly due to SIRS and multiorgan failure ,was greatly reduced in the first phase due to the advances in the intensive care in the management of critical patient, and the development of more sophisticated scoring systems to identify patients with more severe forms of the disease[73].

However deterioration and subsequent mortality in the second phase (after 2 weeks) of severe acute pancreatitis which is mainly due to septic complications i.e. infected pancreatic necrosis, still complicates the picture and the decision to operate would be dependent upon combined clinical , radiological and biochemical parameters .The mortality rate for patients with infected pancreatic necrosis is higher than 20%, and up to 80% of fatal outcomes in acute pancreatitis are due to septic complications. In contrast, mortality for sterile necrosis is low and can usually be successfully treated by a conservative approach, although surgery might be required for late complications or persistent severe pancreatitis[74].Most series documented 100% mortality for patients with infected pancreatic necrosis and multi-organ failure[75].

Our reported post-operative morbidity included pulmonary atelectasis in 3 cases out of 19 cases (15.7%) , wound infection in 2 cases (10.5%), deep vein thrombosis 1 case ,burst abdomen in 1 case and incisional hernia in 1 case.

Conclusions

1. Multidisciplinary approach in the management of acute pancreatitis is highly recommended.
2. Severe forms of acute pancreatitis should be managed in centers with special and sophisticated facilities if available.
3. Surgery should be avoided in the early phases of severe acute pancreatitis except the special occasions of perforations or bleeding.
4. pancreatic pseudocyst is the commonest surgical procedure in complicated severe acute pancreatitis.
5. Pancreatic necrosectomy for infected pancreatic necrosis with or without organ failure is a lifesaving .
6. Intervention in the early phases of pancreatic pseudocyst involution, almost doomed with recurrence.

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